ances in all of its various stages. In its earliest and mildest form, telangiectasia only is seen, and newly formed blood vessels with resultant hyperemia and bleeding appear. This may go on to actual ulcer formation. These ulcers vary in size from one to four centimeters in diameter, and may be deep and crater-like. They are usually covered with considerable necrotic material. If this is wiped off, a tenacious, tough, yellow or silver-gray membrane is found to cover the base. Areas of telangiectasis usually surround the ulcer. A rectovaginal fistula occasionally develops from complete destruction of the wall between the rectum and the vagina.

The onset of these changes varies from a period of two or three weeks to several years following the last application of radium and x-ray. Subjectively these patients complain of atypical sharp, dull or bearing-down rectal pain, hemorrhages of bright red or clotted blood from the rectum, and tenesmus, with increasing desire to go to the toilet.

We feel that these phenomena are not common, are usually mild, and certainly cannot be regarded as an objection to the treatment of carcinoma of the uterus or ovary, or uterine fibroids with radium.

The prognosis of these rectal lesions depends entirely upon the fate of the original extrarectal lesion for which the radium treatment was given. In the great majority of cases (73.8 per cent) this has proved to be carcinoma of the uterine cervix.[1] We have found that if the carcinoma is controlled and proper palliative treatment instituted, the factitial changes and ulcers will usually heal and disappear. This treatment must occasionally be continued over a long period, and sometimes even years elapse before the bleeding disappears.

I wish to report the following case as an example of one of the severest forms of the disorder, where a diagnosis was not made and where this benign entity was treated as a primary rectal malignancy.

REPORT OF CASE

Mrs. C. A. in 1930 complained of bloody vaginal discharges and weight loss of one and one-half years' duration. A diagnosis of carcinoma of the cervix of the uterus was made. Two courses of vaginal radium applications were given along with a series of x-ray treatments. Mild symptoms of rectal irritation followed this treatment but disappeared after a few weeks. She has had no uterine symptoms since the last radium application (July, 1930). Repeated pelvic examinations have failed to disclose any evidence of recurrence. In May of 1931 the patient began to pass small amounts of bright red blood with her stools. She went to a physician who told her that she had "piles," and prescribed a box of suppositories. The bleeding continued and increased in severity. She then was given a course of injection treatments for hemorrhoids, without benefit. In August of 1931 she began to complain of a constant dull pain located about six inches above the anal orifice. At the time of bowel movements this became aggravated and changed into a sharp, colicky, knife-like pain. She began to have constant bearing-down sensations and went to the toilet six to eight times a day. She seemed unable to empty her rectum and usually passed only small quantities of bloody mucus. A proctoscopic examination was done in September, 1931, and a diagnosis of primary carcinoma of the rectum was made. Immediate surgery was deemed necessary because of the extreme amount of discomfort. On September 15, 1931, a colostomy was made. She immediately improved and was sent home to gain strength for a posterior resection of the growth.

The patient was examined by me on November 10, 1931, for the purpose of determining whether the growth was primary in the rectum or an extension from the uterus. A number of indurated ridges around the anal canal were present from the recent injection treatment. A large, indurated, fixed, infiltrating, ulcerated mass was felt on the anterior wall of the rectum, about eight centimeters above the anus. The proctoscope revealed the ulcer to be about four centimeters in diameter. The edges were piled up and it was covered with slimy necrotic material. When the base was swabbed, the characteristic silver gray, tenacious membrane was found to cover the ulcer. Numerous telangiectatic areas, which bled on the slightest trauma, surrounded the ulcer. A diagnosis of factitial proctitis with ulceration was made and palliative treatment instituted. She continued to improve and the colostomy was closed January 15, 1932. A proctoscopic examination on October 3, 1932, revealed the ulcer completely healed. Telangiectasis, however, was still present, and the patient passes a small amount of blood with most of her bowel movements.

COMMENT

This case is an example of the extreme untoward effects of improper diagnosis. Suppositories and injection treatments were used, and served only to cloak the real nature of the disorder. A false diagnosis of rectal carcinoma led to an unnecessary colostomy which had to be closed at a later date.

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PURULENT MENINGITIS

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AS an introduction to a review of recent advances in the therapy of purulent meningitis, we wish to present three uncommon cases of meningitis. These patients were admitted to the Riverside County Hospital during the past five months.

REPORT OF CASES

CASE 1.—E. H., a Mexican girl of four years, was admitted on July 16, 1932, and a diagnosis of tubercular meningitis made. Lumbar puncture on admission revealed a normal pressure and cell count. The following day, spinal fluid pressure was 30 millimeters of mercury without increase in cells. Chest x-ray was diagnosed as bronchopneumonia, and lumbar punctures were discontinued until July 20, when convulsions again occurred. Spinal fluid was then turbid and antimeningococcic serum was given before the laboratory reported both Gram-negative and Gram-positive diplococci. Lumbar taps were continued, but bacteria became more numerous and cells increased to 5,500. The tryptophan test was positive. No tubercle bacilli were found in direct smears. The organisms were identified as *Meningococcus crassus*. The patient died July 24, and permission for autopsy was not obtained. Guinea-pig inoculated with fluid showed tubercles in the liver, spleen, and one kidney.

TABLE 1.—*Case No. 3. Summary.*

Day	Hour	Pressure	Cells	Bact.	Amt. Rem.	Surgery
1	10 p. m.	32 mm.	1200	0 (?)*	10 c.c.	.....
2	8 a. m.	16	760	Rare	20 c.c.	Mastoidectomy
	1 p. m.	14	1282	Few	30 c.c.	Carotid exposed; 10 c.c. acriflavine
	10 p. m.	32	.....	.....	30 c.c.	.....
3	8 a. m.	14	345	Num.	21 c.c.	10 c.c. acriflavine, left carotid
	3 p. m.	26	417	.....	5 c.c.	(Block ?)—Cisternal—bloody
	11 p. m.	14	.....	.....	10 c.c.	.....
4	8 a. m.	22	170	Many	23 c.c.	10 c.c. acriflavine
	3 p. m.	13	550	.....	25 c.c.	.....
	10 p. m.	48	1575	.....	50 c.c.	.....

* Technician not available.

Meningococcus crassus is reported[1] as nonpathogenic, a secondary invader in meningitis caused by other organisms. It is readily identified by cultural methods.

* * *

CASE 2.—G. M., a white man of forty-seven years, was admitted on November 6 with a history of headaches, dizziness, delirium, and coma, which developed during the two days previous to admission. When I first saw him the following day, the patient was in deep coma. Lumbar puncture was done and fluid was found under increased pressure and turbid. Pneumococci were present in enormous numbers. Blood count showed only 5,700 leukocytes with a temperature ranging to 105 degrees. Six hours later 37 cubic centimeters of spinal fluid were removed showing a 50 per cent reduction in cells. At 8 p. m. cisternal and lumbar taps were done simultaneously and normal saline introduced by gravity pressure through the cisternal needle until clear flow was established from the lumbar needle. Condition failed to improve and the patient expired the following morning. At autopsy the entire brain surface was found covered by a thick, greenish exudate.

Obviously, subarachnoid drainage or instillations will have little effect upon such extensive infections which are seen late. While irrigations of the spinal canal will sometimes prevent a low spinal block in stages where the fluid is too thick to flow, it was employed in this case to reduce the number of bacteria present. Kolmer[2] advises against the injection of chemical antiseptics into the subarachnoid space.

* * *

CASE 3.—H. H., a white boy of eight years, was admitted on the night of November 12, 1932, with evidence of a left otitis media and meningitis. Meningeal symptoms apparently developed on November 7. On admission, temperature was 104.4 degrees, with white blood cells 31,600. Lumbar puncture revealed a turbid fluid with a pressure of 32 millimeters of mercury. Because of the history suggestive of otitic origin, no antimeningococcic serum was given. The following morning, left mastoidectomy* was done under ether anesthesia. Lumbar puncture at noon revealed further increase in cells and bacteria. Direct smears from the mastoid cells were negative for bacteria and subsequent cultures were sterile. In view of the sterile mastoid and the positive identification of streptococci in the spinal fluid, the patient was again anesthetized and the left common carotid exposed* by an incision

* Operations by Drs. E. P. Miller and W. K. Templeton.

TABLE 2.—*Summary from Literature, Showing Results of Intracarotid Therapy.*

Series	Staph.	Strept.	Pneumo.	Mening.	Preg'l Iodin	Acriflavine	Serum	Right Carotid	Both Carotids	Recovery	Death
Crawford (10)	3	..	..	..	3	..	..	1	2	2	1
	..	3	..	..	3	..	..	1	2	1	2
	..	..	..	3	3	..	3	..	3	2	1
Dowman	1	..	..	..	1	..	..	1	..	1	..
	..	3	..	..	3	..	..	3	..	1	2
Davis	1*	1*	..	..	1	..	..	..	1	..	1
	..	1	..	..	1	..	..	..	1	..	1
Spurling	1	..	..	..	1	..	..	..	1	..	1
	..	1	..	..	1	..	..	..	1	..	1
Lindemuller	..	1	..	..	1	..	..	1	..	..	1
	..	..	..	1	1	..	..	1	..	..	1
Ersner and Mendell (6)	..	2	..	..	2	2	..	..	2	2	..
Kolmer (2)	..	7	..	..	(5)	(5)	2	..	7	..	7
	..	..	3	..	..	..	(3)	..	(3)	..	3
	..	..	..	1	..	..	1	..	1	1	..
McMahon	..	..	1	..	..	..	1	..	1	1	..
Kauffman	..	1	..	..	1	1	..	..	1	..	1
Total	6	20	4	5	27	8	10	8	26	11	23
Recovery	3	4	1	3	9	2	4	3	8	..	..
Death	3	16	3	2	18	6	6	5	18	..	..

* Mixed infection.

parallel to the anterior border of the left sternomastoid muscle. The wound was left open and a guide ligature placed around the carotid. Ten cubic centimeters of 0.5 per cent neutral acriflavine were injected. This was repeated on November 14 and 15, with spinal fluid findings as shown in the chart. Culture of the fluid showed hemolytic streptococci. Blood culture taken on November 15 remained sterile. At no time was there evidence of acriflavine in the spinal fluid, although a distinct coloration could be seen on the left side of the face. At the end of the third hospital day the child showed a marked change, spinal fluid pressure rising, coma deepening, and breathing irregular. Patient expired the following morning. At autopsy the coloration of the left side of the face was marked, but there was no tinge to the brain or meninges. The brain was wet and edematous, with no organized fibrin or exudate. Summary of condition at different hours is given in Table 1.

This was our first attempt at intracarotid therapy, and we feel justified in interpreting the results as encouraging although the outcome was a fatal termination. We are unable to explain the absence of the dye in the spinal fluid; the urine remained heavily colored. We were unable to obtain Pregl's solution of iodin at this time and resorted to acriflavine. In the absence of continuous spinal drainage, as advocated by Retan,[3] or by surgical means, we believe this case demonstrates the necessity of dehydration by limiting fluids and hypertonic solutions intravenously to accomplish two purposes: (*a*) concentration of serum or antiseptics in the spinal fluid; (*b*) control of cerebral edema and intracranial pressure.

INTRACAROTID THERAPY

The intracarotid treatment of purulent meningitis was first advocated by Kolmer[4] after considerable experimental work on artificially produced pneumococcic meningitis in dogs. By exposing the carotids and injecting anti-pneumococcic serum into both arteries daily for at least three days, he was able to show recoveries in 60 per cent of the animals. Intravenous chemotherapy produced no results in his work. He recommended the procedure for a clinical trial in human cases, admitting it to be a major procedure, theoretically sound and experimentally successful, justifiable in a condition where the mortality approaches 100 per cent.

The technique was first applied, to our knowledge, for the introduction of arsenical preparations in cases of paresis.[5] Since Kolmer's original work, thirty-four cases of purulent meningitis have been reported as treated by this method, with a recovery of 32.3 per cent. It is advisable to use a small tuberculin needle to avoid bleeding from the carotids. Drainage by the lumbar or cisternal route should follow the injection shortly. We do not agree with Ersner and Mendell[6] that partial thrombosis which occurred in one of their patients favored the course of the disease by putting the part at rest. Unimpaired circulation is paramount in an infected field.

The literature on other types of therapy which have effected cures is amply covered by Rosenberg and Nottley[7] and Applebaum.[8] The results indicate that intracarotid therapy offers another tenable method, especially if we accept former statistics by Yerger[9] where the mortality was 97 per cent. Most of the favorable results have been obtained by the use of Pregl's iodin. Kolmer[4] recommends the use of serum or acriflavine and the exposure of both carotids, regardless of the location of the focus of infection.

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MERCURIAL DERMATITIS

FOLLOWING LOCAL APPLICATION OF CREAM FOR REMOVAL OF FRECKLES

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THE scarcity of literature relating to cases of mercurial poisoning, together with its rarity of occurrence in clinical practice in spite of the many thousands of instances in which topical mercury is used, gives us an idea as to its infrequent incidence. In a review of the literature of the past twenty years, I have found fewer than an equal number of cases. Each case, however, has been distinctly different, yet all tend to emphasize two features of hydrargyrism, namely, its varied skin manifestations, and the uncertainty of its degree of involvement and extension.

The most common skin lesion appearing is the papule in the vicinity of the hair follicle, spoken of as folliculitis; but more severe consequences may follow its use, as in the case in which gingivitis, with loss of a tooth permanently, from topical application of mercury for psoriasis, results.[1] There have been no cases reported, however, in which following topical mercury application, the patient suffered dangerous kidney complications. The skin manifestations and types of skin lesions are very numerous, and nearly every type of skin lesion has been described in which mercury has been the responsible aggressor.

REPORT OF CASE

The case to be reported is that of a young woman, age 26, weight 120 pounds, height 5 feet 5 inches, who

1 Beckett, P. E., Dermat. and Syph., (June), 1925.